REMARKS/ARGUMENTS

The Pending Claims

Claims 6, 8-11, 16, 20-22, 47-62, and 67-72 are pending.

Summary of the Amendments to the Claims

Claims 71 and 72 are new and recite similar methods as those of claims 47 and 55, respectively, except that the methods are characterized as *in vitro*. No new matter has been added by way of these amendments to the claims.

Summary of the Office Action

The Office indicates that claims 6-11, 16, 20-22, and 67-70 are allowed.

The Office rejects claims 47-62 under 35 U.S.C. § 112, first paragraph, for allegedly lacking enablement.

Reconsideration of this rejection is hereby requested.

Discussion of the Enablement Rejection

The Office acknowledges that the specification is enabling for inhibiting translation of a Mect1-MAML2 chimeric gene in a cell *in vitro*, but contends that the specification does not provide enablement for inhibiting translation of a Mect1-MAML2 chimeric gene *in vivo*. In particular, the Office contends that the delivery of an effective concentration of a nucleic acid to a target cell, such that a target gene is inhibited to a degree necessary to result in a therapeutic effect, is unpredictable. Therefore, the Office considers that a skilled artisan would require specific guidance to practice the claimed methods *in vivo* in all organisms. The rejection is traversed for the following reasons.

Applicant respectfully submits that the inventive methods are sufficiently enabled by the specification. The specification describes suitable routes by which the inventive compositions can be administered (see, e.g., paragraphs 44, 45, and 49-51). Additionally, the specification describes suitable formulations and dosages for the inventive compositions (see, e.g., paragraphs 41-43, 46-48, and 52). Thus, an ordinarily skilled artisan having the benefit

of the disclosure of the present application would be able to practice the claimed methods without undue experimentation.

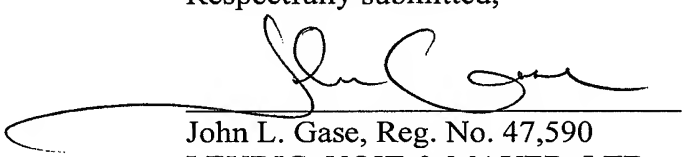
Furthermore, Applicants note that RNAi has been used successfully *in vivo* to inhibit translation of targeted genes. For example, Jang et al. (*Breast Cancer Res., 10(1)*: R11 (Epub 2008); submitted herewith) describes the *in vivo* administration of adenine nucleotide translocator (ANT) 2 shRNA to a mouse tumor model, which led to suppression of ANT2, inhibition of tumor growth, and induction of tumor regression associated with apoptosis (see page 11, paragraph bridging columns 1 and 2). Kong et al. (*Genetic Vaccines and Therapy, 5*: 4 (Epub 2007); submitted herewith) describes the administration of siRNA against the respiratory syncytial virus (RSV)-NSI gene to rats infected with RSV, which led to a reduction in RSV infection and pulmonary pathology (see Abstract). Additionally, there are multiple ongoing clinical trials using RNAi technology, such as Sirna-027 and Cand5 (siRNA against VEGF used for treatment of macular degeneration) and ALN-RSV01 (siRNA against N gene of RSV genome used for treatment of RSV infection).

For the above-described reasons, the inventive methods must be considered to be enabled by the specification and the rejection should be withdrawn.

Conclusion

Applicants respectfully submit that the patent application is in condition for allowance. If, in the opinion of the Examiner, a telephone conference would expedite the prosecution of the subject application, the Examiner is invited to call the undersigned attorney.

Respectfully submitted,



John L. Gase, Reg. No. 47,590
LEYDIG, VOIT & MAYER, LTD.
Two Prudential Plaza, Suite 4900
180 North Stetson Avenue
Chicago, Illinois 60601-6731
(312) 616-5600 (telephone)
(312) 616-5700 (facsimile)

Date: November 26, 2008